The Effects of Race, Residence, and Prenatal Care on the Relationship of Maternal Age to Neonatal Mortality

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Abstract: This population-based study explores whether excessive neonatal mortality rates (NMRs) among infants with teenage mothers are attributable to young maternal age or to a translation of environmental disadvantage into reproductive disadvantage. First births from the 1976–79 linked birth and infant death registers for three states are analyzed. The data set is sufficiently large (305,907 births) to measure maternal age in fine gradations while including several control variables in logit analyses. The associations of racial identification and prenatal care with low birthweight, short gestation, and neonatal mortality overshadow and confound the association between teenage and poor outcome. At every maternal age, higher NMRs are observed for Blacks compared to Whites. The hypothesis that excessive neonatal mortality among Blacks is due to the greater frequency of teenage childbearing among Blacks is refuted. Indeed, unlike White, Black primiparae above age 23 experience higher NMRs than most Black or White teenagers. These results suggest that teenage maternity is not the primary causal agent of all of the problems with which it is associated. (*Am J Public Health* 1986; 76:1416–1421.)

Introduction

Infants of teenage mothers in the United States are more likely to experience pre-term birth, low birthweight, and neonatal death than infants born to older mothers. These observations have led to the belief that adverse pregnancy outcomes among teenage mothers are due to their inherent biological immaturity.[1] However, the infants of teenage mothers in the United States are most likely to be born among socioeconomically disadvantaged populations where women at any age may be victims of environmentally induced risk factors for poor childbearing prognoses. These factors include nutritional inadequacy, excessive stress, life-long medical underservice, inadequate housing and sanitation, and many medical conditions and diseases, both chronic and acute, such as genito-urinary tract infections and hypertension.[2–4] During pregnancy, lack of prenatal care, including high-risk screening, prescription of appropriate therapies for identified problems, and referral to tertiary centers for birth as needed, are further ways that social disadvantage can promote higher risks of neonatal morbidity and mortality.[5,6]

Indeed, in reviewing the literature one finds that in those studies where potentially confounding environmental risk factors are controlled, the teenage mothers in their samples do not exhibit higher rates of specific poor pregnancy outcomes than older mothers.[7–12] It has even been suggested that some of the teen years constitute ages inherently at low obstetrical risk.[8,9,11] These studies have provided important evidence that environmental risk factors can play a key role in the association between teen maternity and poor pregnancy outcome. Yet, it would be premature to draw universal conclusions from these studies. Each suffers from some or all of the following limitations: small sample size;[7,10,11] selective and/or homogeneous sample;[7–11] usage of a 20 year old data set;[8,9] and grouping together ranges of teen ages into study categories,[7,9–12] a practice which could obscure any existing differences between ages.

The current study was designed to avoid these limiting features. Drawing from a large, population-based sample and measuring teenage primarily in single years, it investigates whether the maternal characteristics of racial identification, place of residence, or adequacy of prenatal care confound the relation between maternal age and the adverse pregnancy outcomes of low birthweight, short gestation, and neonatal mortality.

Methods

The data are drawn from the 1976 through 1979 linked birth and infant death certificate registers from the states of Washington, Louisiana, and Tennessee. These data include all first births to Black and White women, as defined by each state, excluding births to women of other or unidentified race, multiple births, and cases where maternal age, county of residence, neonatal birthweight or survival status is missing from the certificates. After exclusions, 95 per cent of all Black and White first births remain in the data set, with a total sample of 305,907 births, of which 2,850 resulted in neonatal deaths.

Observed neonatal mortality rates are calculated by maternal age and race. Rate ratios with 95 per cent confidence limits are estimated according to the Mantel-Haenszel method.[13] The data are also analyzed using log-linear statistical techniques,[14] with the data arrayed in a contingency table wherein dimensions are defined by the categorizations of each variable: maternal age (11–13/14/15/16/17/18/19/20–23/24–26/27–29/30–34/35+), maternal race (White/Black), maternal residence (rural/other), prenatal care (inadequate/other), gestational age (<28 weeks/28–31/32–36/37–42/43+/missing), birthweight (<1001 grams/1001–1500/1501–2000/2001–2500/2501–4000/>4000), and neonatal death (yes/no). The data are limited to first births (i.e., to women with no previous history of still or live births) to avoid the potential confounding effects of parity.

The log-linear program, "Loglin,"[15] is used, performing an iterative proportional fitting procedure to obtain maximum likelihood estimates of effects. Only logit models were fit. The general logit model expresses the natural logarithm of the expected conditional odds of an outcome as:

$$\ln\left(\frac{P_{ij\ldots n}}{1-P_{ij\ldots n}}\right) = \beta + \beta_{1_{(i)}} + \beta_{2_{(j)}} + \ldots + \beta_{N_{(n)}} \qquad (1)$$

$$\text{where } \sum_{k} \beta_{K_{(k)}} = 0 \text{ for } K = 1,2,\ldots,N.$$

1,2,K,...,N refer to the study variables; i,j,k,...,n refer to the categories of each variable. In the models fit, the interactions between the explanatory variables are saturated, i.e., mea-

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sured perfectly, and considered fixed.[16] The summary measures derived from these logit analyses are odds ratios, which well-approximate rate ratios because the odds of death are so small.[17] The purpose of this approach is to describe whether maternal age alters the odds of neonatal death and whether the crude estimate of its effect is confounded by any of the other measured factors.

All of the maternal background characteristics (i.e., race, residence, and prenatal care) may be regarded as proximate variables allowing us to make crude distinctions between childbearing contexts. The term "race" connotes genetic differences between groups, yet as Cooper and David* have argued, races in the United States "are a powerful force in determining health not for biological but for social reasons." Racial identification is a process of social categorization. The social aspect of race is important, given that racial assignments in vital statistics registries can be made on the basis of only one relative's skin color. Accordingly, I have presumed that racial differentials in morbidity and mortality noted by this study are a proxy for environmental disparities related, for example, to socioeconomic status or possibly to regional factors, since the majority of these Black births occurred in southeastern states.

Women who reside in counties that fall outside of Standard Metropolitan Statistical Areas are coded for rural residence. They live in geographically isolated areas that are often medically underserved and remote from medical centers with neonatal intensive care technology. Early fertility may be conventional in remote rural areas, and its relation to poor pregnancy outcomes may be confounded by the socioeconomic and medical disadvantages associated with rural residency.

Prenatal care is defined as "inadequate" according to an algorithm used by Gortmaker[5] and by the Institute of Medicine[18] (the latter being adapted from one originally used by Kessner, *et al*[19]). In effect, this algorithm identifies those women who have either no contact or the least possible interface with the medical care system during their pregnancies. Using certificate data, the content or quality of prenatal care received by those women who fall outside of the inadequate category cannot be determined.

Birthweight is an objective and highly reliable measurement, and predicts neonatal mortality very well. However, some low birthweight (LBW) infants are term babies suffering from growth retardation. These small-for-gestational-age (SGA) infants have better changes of surviving the neonatal period than the same weight babies who are born preterm.[20,21] Among teenage mothers, LBW babies are more likely to be SGA than among older mothers.[4,9] Unfortunately, gestational age is most unreliably measured among infants born to teenage, Black, rural, and other economically disadvantaged women.[22] A "missing gestational age" category is included in this analysis to reduce this bias. Gestational age information is missing for 5.3 per cent of the births in the sample, ranging from a low of 3.7 per cent among White 27–29 year old mothers to a high of 13.5 per cent for White 11–13 year olds.

Results

In Table 1, slightly curvilinear trends are observed in the crude relation between maternal age and neonatal mortality.

TABLE 1—Observed Neonatal Mortality Rates by Maternal Age and Race in Number of Deaths per 1000 Live Births with Rate Ratios (Black/White) n = number of births

Maternal Age (years)	Neonatal Mortality Rates						Rate Ratio (Black/White) with 95% Confidence Interval	
	Overall		Black		White			
	n	*rate*	*n*	*rate*	*n*	*rate*	*RR*	*95% CI*
11–13	641	28.1	448	33.5	193	15.5	2.16	(.64, 7.29)
14	2,697	23.7	1,704	24.1	993	23.3	1.04	(.62, 1.74)
15	8,302	15.3	4,215	18.5	4,087	12.0	1.54	(1.08, 2.19)
16	16,969	15.0	7,035	16.8	9,934	13.8	1.22	(.95, 1.57)
17	24,209	11.4	8,124	14.3	16,085	9.9	1.44	(1.13, 1.83)
18	29,308	10.4	8,679	14.3	20,629	8.7	1.64	(1.31, 2.06)
19	31,561	8.9	7,999	13.3	23,562	7.4	1.79	(1.14, 2.27)
20–23	99,888	8.2	17,914	12.7	81,974	7.3	1.74	(1.49, 2.02)
24–26	49,834	7.3	5,591	16.5	44,243	6.1	2.68	(2.13, 3.36)
27–29	27,158	7.6	2,540	15.0	24,618	6.8	2.19	(1.55, 3.09)
30–34	13,127	8.8	1,179	15.3	11,948	8.1	1.88	(1.15, 3.08)
35+	2,213	8.1	280	14.3	1,933	7.2	1.97	(.66, 5.86)
TOTAL	305,907	9.3	65,708	14.9	240,199	7.8	1.91	(1.77, 2.06)
Age Standardized Rate Ratio (Black/White)							1.68	(1.58, 1.79)

TABLE 2—Neonatal Mortality Rate Ratios, Teen Ages vs Ages 24–26, by Race

Maternal Age (years)	Rate Ratios* with 95% Confidence limits	
	White	Black
11–13	2.54 (.85, 5.35)	2.03 (1.19, 4.92)
14	3.82 (2.56, 37.29)	1.46 (1.02, 2.16)
15	1.97 (1.45, 2.67)	1.12 (.84, 1.50)
16	2.26 (1.85, 2.76)	1.02 (.77, 1.35)
17	1.62 (1.34, 1.97)	.87 (.67, 1.14)
18	1.42 (1.18, 2.37)	.87 (.67, 1.13)
19	1.21 (.99, 1.48)	.81 (.62, 1.84)

*Reference group is maternal age 24–26

This relation holds true overall, and for Blacks and Whites separately, although Blacks exhibit greater uniformity in risk levels between teenage and older mothers. For Blacks, the lowest neonatal mortality rates occur to 19 through 20–23 year olds, while for Whites the lowest rates are observed among mothers in the mid- and late 20s. Furthermore, Blacks experience higher rates of neonatal mortality than Whites at every age. The overall racial differential in neonatal mortality rates drops only trivially (from 7.07 to 6.82 deaths per 1,000 live births) when teenagers are excluded from the analysis.

Racial variation in the relation of maternal age to neonatal mortality is also illustrated in Table 2. Among Whites, all teenagers experience substantial excessive neonatal mortality compared with mothers in their mid-20s, although the size of the rate ratios decreases as teen age increases. Among Blacks, however, only those teenage mothers of 14 years of age or younger experience excessive neonatal mortality compared with mothers in their mid-20s.

Table 3 shows what percentage of each group is Black, received inadequate prenatal care, and resided in rural areas. The distributions of these variables by age take the same slightly u-shape as the distribution of neonatal mortality rates by age. The ages at highest risk of neonatal mortality are also more likely to have the greatest percentage of Blacks, rural residents, and women receiving inadequate prenatal care.

*Cooper R, David R: The concept of race in public health and epidemiology. Paper presented at the 113th Annual Meeting of the American Public Health Association, Washington, DC, 1985.

TABLE 3—Percentage of Mothers by Age Group with Selected Characteristics

Maternal Age (years)	% Black	% Inadequate Prenatal Care			% Rural		
		Overall	White	Black	Overall	White	Black
11–13	70	39	43	37	42	49	38
14	63	36	34	38	37	47	31
15	51	30	29	32	38	46	31
16	42	27	26	29	38	43	31
17	34	24	22	27	39	43	31
18	30	22	20	26	38	41	31
19	25	19	17	23	37	39	30
20–23	18	15	14	20	34	35	29
24–26	11	12	12	18	27	27	22
27–29	9	11	11	17	22	23	17
30–34	9	11	11	18	21	21	20
35+	13	14	13	25	26	27	23

Expected neonatal mortality rates of maternal age under the logit model controlling for the main effects of race, residence, and prenatal care are estimated for each combination of social characteristics (Table 4). Wide variations in rates are evident for any given maternal age according to which social characteristics apply.

Comparing hypothesized logit models clarifies the relation between variables. Each of the three environmental characteristics is associated with neonatal mortality in the predictable directions, and with maternal age. Thus, the unadjusted estimate of the association between maternal age and neonatal mortality is biased. Indeed, when race is controlled, the effects of age on neonatal mortality remain, but are diminished. For example, the unadjusted relative risk of neonatal mortality for 11–13 year olds is 2.5 but drops to 2.0 when adjusted for race. Controlling for prenatal care also reduces the association between maternal age and neonatal mortality. Controlling for residence as measured here has no effect.

Estimated odds ratios for each covariate before and after controlling for each of the other measured factors are given in Table 5. When either gestational age or birthweight is controlled, the effects of maternal age and residence on mortality are completely attenuated. The effects of prenatal care drop substantially from an unadjusted odds ratio of 1.9 (inadequate/other) to 1.32 and 1.17 when controlling for birthweight or gestation, respectively. The apparent disadvantage of Black racial identification is attenuated when gestational age is controlled. After adjusting for birthweight, the effects of race change direction, and the disadvantage is then experienced by White infants. The unadjusted odds ratio (Black/White) is 1.89, while the odds ratio when birthweight is controlled is .92.

Up to this point, birthweight and gestation have been included in logit models as explanatory variables, with neonatal mortality being the response variable. The results so far imply differential birthweight and gestational age distributions by maternal age, race, residence, and prenatal care. To clarify the associations between maternal age and birthweight and gestation, analyses are also performed where birthweight or gestational age is the dependent variable, with maternal age, race, residence, and prenatal care acting as explanatory variables.

Each of these covariates is associated with gestational age, independent of the others. However, the estimates of the effects of young maternal teenage on gestation are unstable, changing greatly when other factors are controlled. For example, the unadjusted odds ratios (11–13 year olds/24–26 year olds) of gestations of <28 weeks, 28–31 weeks, or none reported on the birth certificate ("missing" gestation) are 12.0, 10.0, and 1.22, respectively. After adjustment for the effects of race, these odds ratios change to 5.23, 5.54, and 1.94. In addition, women of 35+ years exhibit higher risks of very short gestations than do women in their 20s and early 30s, but not as high as those experienced by the youngest teenagers. Unadjusted odds ratios (35+/24–26) of <28 week, 28–31 week, and missing gestations are 2.97, 1.52, and .80. When adjustments are made for the environmental variables, the risks of very short gestations are reduced for the 11–13 year olds, but remain the same or increase slightly for the 35+ age group.

The missing gestation category is the primary link between residence and gestation. While the estimates of the effects of residence on gestation are small on all other gestational age categories, rural residence is moderately

TABLE 4—Expected Neonatal Mortality Rates by Maternal Age for Various Combinations of Maternal Characteristics Estimated under the Logit Model Controlling for the Main Effects on Mortality of Maternal Age, Race, Residence, and Prenatal Care

Maternal Age (years)	NEONATAL MORTALITY RATES							
	White Other Residence Other Care	Black Other Residence Other Care	White Rural Residence Other Care	Black Rural Residence Other Care	White Other Residence Inadequate Care	Black Other Residence Inadequate Care	White Rural Residence Inadequate Care	White Rural Residence Inadequate Care
11–13	14.9	26.8	16.9	28.4	26.8	42.4	28.4	47.8
14	13.0	21.9	14.7	24.7	21.9	36.9	24.7	41.6
15	9.1	15.3	10.3	17.2	15.3	25.7	17.2	29.0
16	9.7	16.3	10.9	18.3	16.3	27.3	18.3	30.8
17	7.8	13.0	8.7	14.7	13.0	21.9	14.7	24.7
18	7.2	12.0	8.1	13.6	12.0	20.2	13.6	22.8
19	6.5	10.9	7.3	12.3	10.9	18.3	12.3	20.7
20–23	6.5	10.9	7.3	12.3	10.9	18.3	12.3	20.7
24–26	6.1	10.3	6.9	11.6	10.3	17.3	11.6	19.5
27–29	6.5	10.9	7.3	12.3	10.9	18.3	12.3	20.7
30–34	7.5	12.5	8.4	14.1	12.5	21.1	14.1	23.8
35+	6.6	11.1	7.5	12.5	11.1	18.7	12.5	21.2

TABLE 5—Estimated Odds Ratios of Neonatal Mortality by Maternal and Infant Characteristics before and after Adjusting for the Effects of Other Factors

Variables	Crude	Odds Ratios Adjusting for the Effects of:						
		Age	Residence	Prenatal Care	Race	Birthweight	Gestation	All
Age (years)								
11–13	3.98		3.90	3.32	2.84	1.47	.94	1.33
14	3.33		3.33	2.83	2.47	1.39	.92	1.25
15	2.10		2.10	1.90	1.69	1.05	.85	.98
16	2.10		2.06	1.90	1.72	1.23	.94	1.13
17	1.59		1.56	1.46	1.38	1.03	.91	.98
18	1.44		1.44	1.35	1.27	1.09	.89	1.04
19	1.22		1.22	1.17	1.11	1.13	.92	1.06
20–23	1.13		1.13	1.11	1.09	1.11	1.00	1.11
24–26	1.00		1.00	1.00	1.00	1.00	1.00	1.00
27–29	1.04		1.06	1.04	1.04	1.09	1.06	1.11
30–34	1.20		1.22	1.22	1.22	1.16	1.15	.94
35+	1.13		1.13	1.11	1.11	.79	.77	.74
Race								
White	1.00	1.00	1.00	1.00		1.00	1.00	1.00
Black	1.89	1.68	1.98	1.82		.92	.96	.86
Residence								
Rural	1.12	1.12		1.12	1.17	1.12	1.04	1.08
Other	1.00	1.00		1.00	1.00	1.00	1.00	1.00
Prenatal Care								
Inadequate	1.92	1.75	1.90		1.75	1.32	1.17	1.27
Other	1.00	1.00	1.00		1.00	1.00	1.00	1.00
Gestation (weeks)								
<28	276.66	288.13	276.66	271.13	276.66	5.13		5.12
28–31	49.54	51.59	49.54	49.53	49.54	2.35		2.30
32–36	5.95	6.07	5.95	5.83	5.95	1.71		1.74
37–42	1.00	1.00	1.00	1.00	1.00	1.00		1.00
43+	1.65	1.69	1.65	1.65	1.65	1.71		1.74
missing	8.35	8.36	8.35	7.71	8.35	2.60		2.35
Birthweight (grams)								
<1001	1017.09	999.56	1017.09	999.56	1037.64		366.90	378.77
1001–1500	119.67	115.27	119.67	115.27	119.67		58.27	60.16
1501–2000	26.17	25.72	26.17	25.72	26.70		17.20	17.41
2001–2500	15.87	15.60	15.87	15.60	15.87		5.39	5.46
2501–4000	1.00	1.00	1.00	1.00	1.00		1.00	1.00
4000+	1.38	1.39	1.38	1.38	1.38		1.36	1.35

Estimated odds ratios are derived from logit models, with crude figures being the unadjusted estimates. The standard category used for each variable is: maternal age (24–26); residence (other); prenatal care (other); race (White); birth weight (2501–4000 gms); gestation (37–42 weeks).

associated with missing gestational age (odds ratio, rural/other = 2.51). This estimate remains unchanged when race or maternal age is controlled, but is reduced to 2.0 when adjusted for prenatal care. Because missing gestation and inadequate prenatal care are positively associated, missing gestational age may be another indicator of inadequate prenatal care.

The main effects of maternal age, race, and prenatal care are associated with birthweight, but residence is not. Adjustment for race reduces the risk of low birthweight among teenagers and increases it for older first time mothers. For instance, the unadjusted odds ratio (14/24–26 year olds) for birthweight of 1001–1500 grams is 3.74, but decreases to 2.55 when adjusted for race.

The crude association of racial identification and birthweight is not confounded by maternal age, prenatal care, or residence. As has been documented, Black Americans experience higher rates of low birthweight (less than 2500 grams) than Whites.[18] In these data, the disparity in risk between Blacks and Whites is greater in the very low birthweight categories (<1001 grams, 1001–1500 grams) than in the other low birthweight categories. The relative odds, Black versus White, of bearing low birthweight infants are 3.74, 2.61, 2.13, and 1.98 for the respective weight groups of <1001 grams, 1001–1500 grams, 1501–2000 grams, and 2001–2500 grams.

The effects of prenatal care on low birthweight also are unchanged by controlling for the other covariates, although the magnitude of its effect is not as great as that of race. While the odds ratio (Black/White) of 1001–1500 grams birthweight is 2.61, the respective odds ratio for prenatal care (inadequate/other) is 1.49. The effect of race on birthweight is so much greater than that of prenatal care that, in this data set, Blacks in the *preferred* prenatal care group remain at increased risk, exhibiting, for example, twice the relative risk of bearing very low birthweight infants as Whites with *inadequate* prenatal care.

Discussion

The results of this study indicate that the neonatal risks associated with teenage maternity are not uniform. They vary by teen age, by prenatal care, and, most prominently, by racial identification. In terms of biologic versus environmental causes of excessive risk among teenagers, the results for the youngest teenagers are inconclusive. If the crude distribution of neonatal mortality rates by maternal age had not been confounded, its slightly curvilinear shape would have suggested that biological disadvantage is experienced by

those who have recently achieved menarche and those approaching menopause. Given the evidence of confounding of the age/mortality association, a prima facie acceptance of the biological explanation seems unwarranted.

The confounding of the maternal age/neonatal mortality distribution may be related to the social selection process regulating age at first birth. In the United States, socioeconomically advantaged teenagers, who have enjoyed life circumstances conducive to healthy and unimpaired physical growth and development rarely bear children. (Although increasing proportions of advantaged teenagers are sexually active, the majority of pregnancies that occur among this group are terminated.) Disadvantaged Americans, on the other hand, often initiate childbearing by their late teens and currently account for the majority of teenage births. Such social selection is consistent with results of this study showing that the teenagers at highest risk of adverse pregnancy outcomes, specifically those under 17 years old, are more likely to be Black, to live in rural areas (especially if White), and to receive inadequate prenatal care (especially if White) than older first time mothers. This suggests the possibility that even their excessive rates of short gestation, low birthweight, and neonatal mortality may result from a variety of physiological consequences of their environmental disadvantage, not primarily from inherent and intractable biological developmental limits.

In addition, when interpreting the high risks exhibited by the youngest teenagers, several cautions apply. The risk estimates for the 11–13 and 14 year olds, while the highest, are subject to large sampling variability. A number of reporting errors is plausible. As Garn and Petzold have noted, such early fertility implies early menarche, which itself is associated with short stature, an independent risk factor for poor neonatal outcome.[8] The population of 11–13 and 14 year old mothers is socially selected, as well. Sexual activity is still quite atypical in the United States at such early ages,[23] prompting speculation that very peculiar social circumstances (extreme isolation from the social and economic mainstream, or even such possibilities as rape or incest) may account for these pregnancies and births, and, perhaps, influence their outcomes adversely. As maternal age increases beyond the early 20s, a selection process would also account for the older mothers in the data set, but exactly how it operates defies conclusive interpretation. Some women choose to postpone their first births because educational and professional goals supervene in their late teens, 20s, and even early 30s. Other women experiencing their first birth at older ages do so not by choice, but because sub-fecundity has interfered with their achieving motherhood earlier. The occurrence of either of these selection processes could bias the results. Depending on which process prevailed, the risks to teenage relative to older primiparae, overall and within race, could be either over- or underestimated. Since fertility impairment is uncommon and its representation in the data set is reduced (especially at the older ages) by the exclusion of women with previous stillbirths and, implicitly, of those who are infertile, careful consideration suggests that the social selection process would predominate at the older ages. If social selectivity were operative, the relative risks of mortality for the youngest teenagers (by virtue of their being relative to the risks for older, more advantaged, primiparae) would be inflated.

The excessive neonatal mortality risks exhibited by teenagers are mediated by gestational age and birthweight. While it may be that such excessive neonatal morbidity signals biological immaturity, this is at best only a partial explanation. For example, the increased risks of short gestation, low birthweight, and neonatal mortality among teenagers are all reduced when controlling for race or prenatal care. Among teens <15, for example, grossly inadequate prenatal care is associated with almost one-third of their total neonatal deaths. Also, the relation between young teen age and gestational age is in part a function of a larger proportion of infants with missing information on gestational age among the youngest teenagers than among older mothers. Missing information on gestational age is not a biologic phenomenon.

The large and persistent racial differentials in neonatal mortality risk observed at all ages offer a valuable response to the question of whether teen age of mother contributes uniquely or importantly to neonatal mortality risk beyond its association with environmental disadvantage. In this sample, trends in neonatal mortality by maternal age follow different paths according to race. Above age 14 among Blacks, neonatal mortality rates by age are remarkable for their relative uniformity and for their lage size in comparison with White rates. Black primiparae in their mid-20s, late 20s, and 30s have rates of short gestation, low birthweight, and neonatal mortality that are *higher* than those exhibited by White teenagers above age 14 and Black teenagers above age 16. This finding is striking given the fair assumption that, unlike teenage mothers of either race, older Black mothers represent, in part, a share who are socioeconomically, educationally, and professionally advantaged.

In addition, if none of the teenage pregnancies in this data set had occurred, the racial disparity in neonatal mortality rates would have dropped only trivially. This finding contradicts the view that higher Black neonatal mortality rates compared with White are attributable to the greater incidence of teenage childbearing among Blacks. It further suggests that bridging the racial gap in neonatal mortality experience may not be achieved by teenage pregnancy prevention alone.

In sum, this population-based study demonstrates that the associations between teenage maternity and each of the adverse pregnancy outcomes of preterm birth, low birthweight, and neonatal death are confounded. The confounding may reflect environmentally induced risk factors as the likely agents of excessive rates of these outcomes among infants of teenage primiparae.

ACKNOWLEDGMENTS

The author was supported in this work by fellowships from the American Association of University Women Educational Foundation and from the National Institute of Mental Health, grant #5T32MH14246. This research was also supported in part by grant #05146 from the Robert Wood Johnson Foundation. I am grateful to Steven Gortmaker, Beatrix Hamburg, Robert LeVine, Marcello Pagano, Roger Banks, and Rachel Snow for providing valuable comments; and to Arthur Sobol for computer programming assistance.

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Prudent Lifestyle for Children: AAP Policy Statement on Dietary Fat and Cholesterol

The American Academy of Pediatrics' (AAP) Committee on Nutrition, in a newly-released statement on children's diets appearing in the September issue of *Pediatrics*, contends that there is no "compelling new evidence to make recommendations concerning modification of the diet during the first two decades of life." However, the Committee advises that since diet is only one factor affecting the risk of coronary heart disease and atherosclerosis, health professionals should recognize that obesity, activity patterns, hypertension, and cigarette smoking (and possibly smokeless tobacco) are additional important contributing factors, some of which the individual has control over.

Updating a 1983 statement, the Committee continues to recommend that "diets that avoid extremes are safe for children for whom there is no evidence of special vulnerability," adding that, "It would seem prudent not to recommend changes in current dietary patterns without first assessing the effects on growth, development, and such measures of nutritional adequacy as the status of iron."

Recently, the American Heart Association, the American Health Foundation, and a consensus development panel sponsored by the National Institutes of Health (NIH) recommended reducing fat and cholesterol intake with the intention of preventing the onset of coronary heart disease in adulthood. The Committee, however, questions whether the diet proposed by the NIH panel will be effective in decreasing cholesterol levels during the first two decades of life, or that the diet will adequately support growth, especially during the adolescent growth spurt. "The proposed changes would affect consumption of foods currently providing high quality protein, iron, calcium, and other minerals essential for growth," the Committee says.

The AAP's guidelines are less strict than those from the NIH panel, but the two groups are not significantly different in their recommendations. The complete committee statement text is available on request from the American Academy of Pediatrics, 141 Northwest Point Road, P.O. Box 927, Elk Grove Village, IL 60007.